# CLINICAL PROCEEDINGS

*of the*

## CHILDREN'S HOSPITAL

WASHINGTON, D. C.



*February 1954*

VOLUME X NUMBER 2

# CLINICAL PROCEEDINGS

## OF THE CHILDREN'S HOSPITAL

13th and W Streets, Washington 9, D. C.

Vol. X *February 1954* No. 2

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PUBLISHED MONTHLY BY THE STAFF AND RESEARCH FOUNDATION OF THE CHILDREN'S HOSPITAL, WASHINGTON, D. C.

Cases are selected from the weekly conferences held each Sunday morning at 11:00 A.M., from the Clinicopathological conferences held every other Tuesday afternoon at 1:00 P.M., and from the monthly Staff meeting.

This bulletin is printed for the benefit of the present and former members of the Attending and Resident Staffs, and the clinical clerks of Georgetown and George Washington Universities.

Subscription rate is $2.00 per year. Those interested make checks payable to "Clinical Proceedings Dept.," The Children's Hospital, Washington, D. C. Please notify on change of address.



Entered as second class matter November 21, 1946 at the post office at Washington, D. C., under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 538, Act of February 28, 1925, authorized January 17, 1947.

# KLEBSIELLA PNEUMONIA TREATED WITH TETRACYCLINE (ACHROMYCIN)

## A Case Presentation

Milton Glatt, M.D.

T. H.,* a four and a half year old white male was admitted to Children's Hospital on October 7, 1953 with the chief complaints of fever, abdominal pain, vomiting and shaking chills. The patient apparently was well until one week prior to admission when he was treated for what the mother said was "asthma." He made an uneventful recovery and was well until the day preceding admission, at which time he began running a fever. On the day of admission he awoke crying and complaining of abdominal pain. He vomited once and had a shaking chill. The remainder of the history was noncontributory.

Physical examination on admission revealed a well developed, well nourished four and a half year old white male lying flat in bed in no apparent acute distress. The temperature was 103 degrees farenheit rectally; pulse 110 and respiration 20. The lungs were clear to percussion and auscultation. There was a soft systolic murmur heard best in the third intracostal space to the left of the sternum. The abdomen was soft and nontender with some voluntary spasm. Normal bowel sounds were heard throughout. The remainder of the examination was not remarkable.

On admission the white count was 37,100 with 90 per cent neutrophiles. The urine was normal. A nasopharyngeal culture grew out *Klebsiella pneumoniae*. Radiographic examination of the chest revealed an infiltrative area in the left lung field radiating from the hilum out to the parenchyma.

The patient was placed on Tetracycline (Achromycin) the dose being 100 milligrams every six hours. The temperature came down to normal within 48 hours after therapy was initiated. There was gradual clinical and radiological improvement until discharge on the seventh hospital day. Subsequent x-rays of the lungs showed complete clearing and the white count and sedimentation rates were normal. The patient has remained asymptomatic since discharge from the hospital.

### DISCUSSION

*Klebsiella pneumoniae* (pneumobacillus, *Bacterium friedlanderi*, *Bacillus mucusus capsulatus*, Friedlander's bacillus) are short, nonmotile, nonsporing, gram negative bacilli which characteristically possess a large capsule. They produce large mucoid colonies on agar media and ferment a number of carbohydrates with the production of acid and gas.[1] They are found in the nose, mouth, and intestinal tract of normal persons. Bloomfield [2] reports an incidence of from 1–5 per cent in the respiratory tract of normal individuals.

Originally discovered in 1883 by Friedlander in the lungs of patients dying with pneumonia, it was believed by him to be the major cause of pneumonias. This misconception was soon corrected, and a study of the

* From the service of Dr. Edward Lewis.

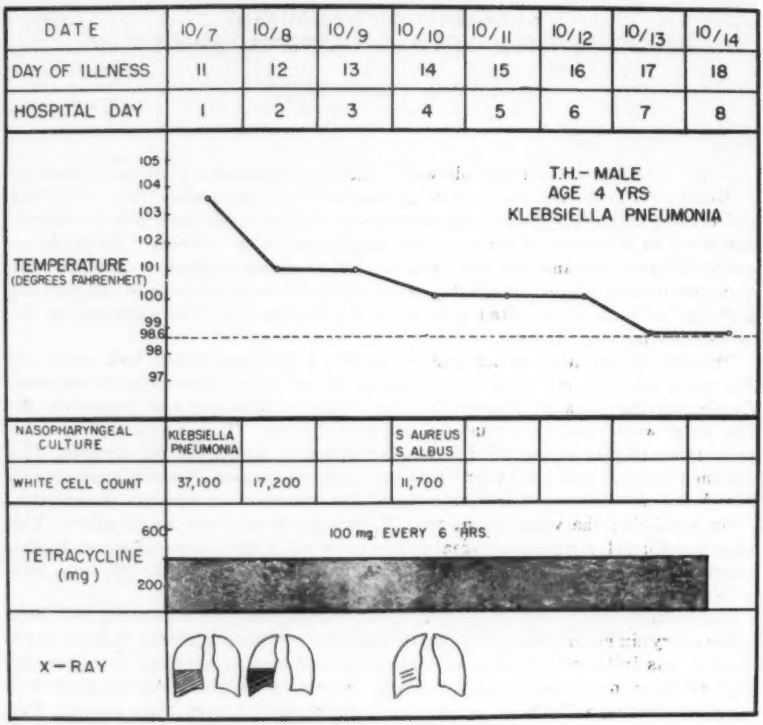


FIG. 1

statistics available in the literature indicates that *Klebsiella pneumoniae* is responsible for only a small proportion ranging from 0.5 to 13 per cent of the total pneumonias encountered. A composite study of the statistics published by various authors dating from 1886 to 1937 and quoted by Julianelle[3] reveals that of a total of 17,260 cases of pneumonia reported, 196 cases or one per cent were ascribed to *Klebsiella pneumoniae*. In their series of 2,450 cases of pneumonia, Perleman and Bullawa[4] report 37 cases or 1.5 per cent to be due to Klebsiella pneumoniae. Heffron[5] reports 0.5 per cent out of a total of 3,319 cases of pneumonia to have been due to *Klebsiella pneumoniae*. Bhatbager and Singh[6] reported 13 per cent of the 100 cases of pneumonia to have had Friedlander's bacillus as their etiologic agent.

The disease is most commonly encountered in adult life and next most frequently during middle age or even later with a tendency to predominate

in males. The predisposing influence of alcoholism has been stressed. Julianelle[3] in 1941 had never encountered an example of this pulmonary disease in infants or children and the infrequency with which this finding is reported in the literature clearly attests to its rarity in the lower age groups. Miller, Orris, and Taus[7] in 1947 reported a case of Friedlander's pneumonia in a newborn. In their review of the literature they were able to find only four reports incriminating *Klebsiella pneumoniae* as the etiologic agent in pneumonia in children. In 1949 Grotts[8] published a series of seven cases of pneumonia in children in which *Klebsiella pneumoniae* was isolated as the sole or predominating agent from nasopharyngeal smears and cultures. The low incidence of this disease as encountered in the pediatric age group was counterbalanced by a high mortality prior to the advent of streptomycin and the broad spectrum antibiotics. The mortality was three to four times that seen in pneumococcal pneumonia and has been reported as having been upwards of 80 per cent. It therefore becomes apparent that the importance of this disease is not in its frequency, but in its mortality. There is early evidence of necrosis followed by liquification, cavitation and even fibrous formation which may leave permanent evidence of past infection. The course of the disease in untreated cases may lead to early death, to clinically suppurative pulmonary disease, or to recovery in a relatively small percentage of cases. The diagnosis is made by recovering the organism from nasopharyngeal smears and cultures.

With the advent of streptomycin as a therapeutic agent the prognosis of this disease has taken a turn for the better. Heilman[9] reported in vivo and in vitro experiments on the effects of streptomycin on the Friedlander group of organisms from which he concluded that it may be useful in the treatment of infections caused by these organisms. Bishop and Rasmussin[10] were the first to report a successful cure using streptomycin in a 59 year old woman in 1945. In 1947 Miller et al[7] were the first to report a similar success with streptomycin in the treatment of Friedlander's pneumonia in a newborn child following the failure of both sulfadiazine and penicillin to control the infection. Following this, Grotts[8] presented seven cases of Friedlander's pneumonia in infants all of which failed to respond to penicillin and sulfadiazine. These same patients showed rapid improvement when treated with streptomycin.

Gruber et al[11] presented the first case of Friedlander's pneumonia with multiple lung abscesses in a thirteen month old boy where an uneventful recovery was made following the use of streptomycin and aureomycin. Kirby and Coleman[12] studied the effects of various antibiotics in eleven patients with Friedlander's pneumonia, and they concluded that aureomycin is probably as effective as streptomycin in the treatment of this infection.

Recently a new drug, Tetracycline (Achromycin) has been made available

for clinical and experimental evaluation. Several recent preliminary reports have indicated that Tetracycline shows characteristics generally similar to aureomycin and terramycin pharmacologically and clinically. In view of the broad similarities and minor differences between these broad spectrum antibiotics it was felt that similar clinical results could be anticipated. The preceding is the first case reported of Friedlander's pneumonia in a child successfully treated with Tetracycline. The inordinately high white cell count, the positive nasopharyngeal culture for *Klebsiella pneumoniae*, as well as radiographic evidence of pneumonia were considered to be presumptive evidence in diagnosing the disease as a Friedlander's pneumonia.

SUMMARY

A brief review and description of Friedlander's pneumonia is presented with special attention to the pediatric age group. A case of Friedlander's pneumonia in a four and a half year old boy is also presented which represents the first such case reported successfully treated with Tetracycline (Achromycin).

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## THE PROBLEM OF THE ABANDONED BABY

J. Peter Martin*

The words "Newborn Infant" are used many times each day. They are general words of descriptive application, such as "Bobbysoxer" or "Housewife." Such terms paint in our minds a picture which seems on the surface

* Medical student, third year, George Washington University School of Medicine.

quite complete, but to which color and warmth may be added by further qualification. We may then refer to this newborn infant as a "well baby" and so achieve such ends. Our picture is now complete, but remains, as yet, unhung, for what in reality is a "well baby?" Is it a person, a state, or a combination of the two? Is it no more than a very small individual free of disease and deformity? Does it infer loving parents? or, for that matter, does it infer parents at all? With these questions in mind, I should like to present the case of L. M.—well baby.

This case is not a medical one in the true sense of the word, but rather a sociological problem, for L. M. is an abandoned baby. Fortunately, abandonments are no longer commonplace in Washington, and with each occurrence, public attention is focused on the problem in the short time it takes for the next newspaper edition to reach the streets. All too frequently, however, the self-same baby whose picture and story drew the sympathetic sighs of millions is forgotten—reabandoned, so to speak—with equal rapidity. Here, even more than in problems medical, the prognosis in the case at hand will be determined by the institution of proper and sufficient therapy. A report on the facilities available and methods used in the dispensation of such cases in the District of Columbia follows the presentation of the case findings.

Patient: L. M. # E-13033.

Chief Complaint: Abandoned baby.

Present Illness: This newborn negro female was found on August 21st, 1953 by a trash collector, who noticed something moving in a paper bag among the refuse from a can which he had emptied into his truck. She was taken to Children's Hospital by a police officer, and admitted on August 21, 1953 as an abandoned baby. The umbilical cord was tied immediately at Children's Hospital. The infant was kept there until her transfer to the District of Columbia General Hospital on August 25th 1953.

Past History: Birth weight (weight when first observed) six pounds, eight ounces. Weight on admission to District General Hospital, five pounds, thirteen ounces. Moderate cyanosis was reported on August 21, 1953. Feedings from August 21 to August 25 was four ounces evaporated milk 1:2 every four hours. At District General Hospital the formula was *"OB # 2."*

Family History: Not available.

Social Status: Abandoned baby.

Physical Examination: August 29, 1953. Age: 8 days. Temperature: 98.6; pulse: 48; respiration: 44. Weight: 6½. Length: 48 cm. Head: 33½ cm.; Chest: 31 cm.

General Appearance: A well-developed, well-nourished, colored female, sleeping peacefully. The appearance: well baby.

Since admission the baby was fed and slept well, and probably will remain in this hospital until arrangements can be made to place her in a foster home.

The case of L. M. is remarkable in but one respect, that being her good fortune in surviving, without ill effects from a treatment which was surely designed to bring about her death. In the great majority of such cases, irreversible physical deformities are apparent either as the cause or effect of abandonment.

Since 1945, the District of Columbia General Hospital has averaged six to eight such admissions each year. These figures may be assumed to represent the total number of abandoned babies found alive in the Metropolitan area, since all such cases are referred to this hospital. Approximately seventy per cent of the cases observed during this period have been negro infants.

At this hospital, the infant is examined and observed until it reaches three months of age. At this time a decision is made concerning its qualifications for placement in a foster home. Investigations of such homes are conducted periodically by the Board of Public Welfare, which pays subsistence totalling $60.00 per month for infants under one year of age and $40.00 per month thereafter to approved foster parents, for the care of each child. If, by the sixth month, the infant has not been declared fit for placement, or if qualified but as yet unplaced, the child is transferred to Junior Village, the government supported children's home for the District of Columbia.

While at Junior Village, the infant is kept in the nursery until it reaches the age of two and a half years. Constant medical attention is afforded the child here by a full-time nursing staff and an attending physician. During the first year of his stay at Junior Village, every effort is made to contact the parents or other relatives to whom the child may be released if wanted, upon approval of the Board of Public Welfare. If parents or relatives are located, following placement in a foster home, the child is recalled and returned, if substantial proof is demonstrated of a desire to care properly for the infant. Due to the predominance of Negroes among foundlings, placement for adoption is not as frequent as might be desired. For the same reason, foster homes are largely established in rural areas where larger families are tolerated more readily. If placement has not been accomplished by the time the child reaches school age, he is enrolled in the school on the grounds of the home where six full-time teachers serve to afford ample educational opportunity. Recreational facilities are abundant, and periodic trips to museums, concerts, the zoo, etc. are scheduled.

The population of Junior Village runs slightly under its capacity of two

hundred children. The average stay per child at the home is approximately three months. The total admissions to the home for the year 1952 were 627, of which some 72 per cent were Negro children. Foundling admissions constitute less than one per cent of the total in any given year, and their stay is for approximately the average of three months reported for all admissions. The major causes of admission to Junior Village are as follows: 18 per cent because of inadequate housing, 13 per cent are due to neglect, and 7 per cent because of alcoholic parents.

Although there are eight licensed adoption placement agencies in the District of Columbia, all but Junior Village and the Board of Public Welfare are operated by church or private organizations, which receive children for placement on a voluntary basis. The most common cause of such committals is the inability of the parent or parents to support their offspring. On the other hand, committals to Junior Village are mostly involuntary. 49 per cent have been taken from their homes and referred by the Women's Bureau of the Police Department. 14 per cent of the children at Junior Village have been placed there by order of the Juvenile Court. The remaining 39 per cent have been domiciled in the home by the Department of Child Welfare of the Board of Public Welfare. Only in a few instances are even these admissions voluntary.

Notwithstanding recent expansion and improvement of facilities at Junior Village, the outlook for the L. M.'s of today is not a pleasant one. The adoption rate has dropped to an all time low, and there are no signs to indicate that it will not drop still further. A dim note is cast as well by the relatively high percentage of returns to the home of children placed for adoption or in foster homes. Ten years ago the return ratio over a ten year period after release from Junior Village stood a little better than 1:1, or 56 per cent returnees and as of 1952, these figures had been improved to 1:2, or 32 per cent returnees. Even these encouraging figures are exceeded by the decreased adoption rate over the same ten years.

In conclusion let us simply say that the outlook for the future of our well baby must be poor, and we hope that she will have the good fortune, which she deserves, to be received into the sort of home where our necessarily fatalistic outlook might prove incorrect. That is a home where human life and human feelings are valued.

NOTE

Data used in the compilation of this paper was obtained by consultation with members of the Social Service Staff of the District General Hospital and the Directors of the Department of Child Welfare, the Washington Home for Foundlings, and Junior Village.

# ACUTE RHEUMATIC CARDITIS

A Case Presented by

Milton Glatt, M.D.

O. H., a ten year old white girl was admitted to Children's Hospital on the night of October 21, 1953 because of an unremitting fever of approximately two weeks duration. The patient apparently was well until sixteen days prior to admission when she began to complain of a sore throat and headache, accompanied by a fever. The patient was treated with penicillin by her physician and the symptoms disappeared but elevation of temperature persisted up to the time of her admission, aureomycin having been used also. There was at no time any precordial or abdominal pain, joint swelling or tenderness, cough or diarrhea. At the time of her admission, the remainder of the history was negative and noncontributory. Several days after admission the mother disclosed the fact that the patient had been seen at the Georgetown University Hospital on July 21, 1951 because of difficulty in breathing and for a chronic headache. At that time the mother was asked about rheumatic fever. No definite history was elicited.

At the out-patient department of Georgetown University Hospital an x-ray of the chest was interpreted as evidence of histoplasmosis. A grade one apical systolic murmur radiating to the left axilla and a grade two aortic diastolic murmur, heard best in the third intercostal space on the left was heard. No notation of rheumatic fever was made and although the patient was scheduled for a subsequent study, she never returned. No further information is therefore available. The family history reveals that one sibling had respiratory distress which was not clearly defined and that the father is a cardiac patient being treated with digitalis.

Physical examination: The patient was a well developed, well nourished pallid ten year old white female in no apparent acute distress. The temperature was 103 degrees Farenheit, pulse was 132 per minute, respirations were 18 per minute, blood pressure was 100/60. There were no palpably enlarged lymph nodes nor were there any subcutaneous nodules found. The neck veins were not engorged and the liver was not palpably enlarged. No sacral edema was present. The positive findings were primarily limited to the examination of the heart which revealed the following:

The PMI was palpable in the left sixth intercostal space just outside of the midclavicular line. It was forceful but no thrill was felt. A blowing, grade two systolic murmur was heard at the apex and radiated to the left axilla.

The quality of the heart sounds was poor and distant. P-2 was greater than A-2. No gallop rhythm was heard. The femoral pulse was easily palpable bilaterally. An electrocardiogram obtained immediately after admission was interpreted as being within normal limits. Fluoroscopic examination of the thorax suggested the presence of an enlarged thymus. The hemoglobin was nine grams, VPC 32 per cent, white blood count 16,400 with a differential of 68 per cent polymorphonuclears, 30 per cent lymphocytes and 2 per cent monocytes. Corrected sedimentation rate was 40 mm an hour. Urinalyses were not remarkable. X-ray of the chest failed to reveal any abnormality other than areas of calcification in both lung fields. Three blood cultures and a throat culture were taken at the time of admission and were subsequently reported as showing no growth. PPD was negative and histoplasmosis skin test was positive. The impression following the entire work up and in keeping with the history and physical findings was active rheumatic heart disease. The heart was not enlarged,

there was mitral insufficiency and mitral stenosis and regular sinus rhythm. The day following admission a pericardial friction rub was heard.

On the recommendation of the cardiac consultant, the patient was placed on cortisone therapy. Three hundred mg were given on the first day, 200 mg on the second day, 100 mg the next two days and a daily maintenance dose of 75 mg was given thereafter. During the same time, penicillin 600,000 units a day, potassium, iron, and sedatives were given. On this regimen the patient failed to respond clinically, the temperature remained elevated, pulse rate was rapid and there was no essential change in the cardiac findings. Cortisone was increased to 125 mg per day on 29 October. On November 3, two weeks after it was first given, the drug was discontinued and the patient placed on aspirin, 15 grains every four hours. On 5 November, a transfusion of 125 cc of packed red cells was given. On 7 November, the liver was palpably enlarged, the neck veins were distended and bilateral basal rales were heard. In keeping with the diagnosis of cardiac failure, the patient was given mercuhydrin and placed in oxygen. Five days later, on November 12, the patient was digitalized with cedilanid and has been on digitalis leaf maintenance therapy ever since. The patient is now slowly recovering.

DISCUSSION

Bernard J. Walsh, M.D.

On the second day after her admission we were asked to see this girl, and we were impressed immediately with the fact that she was very ill due to severe rheumatic fever. Her appearance indicated an overwhelming infection. Detailed examination revealed no rash, subcutaneous nodules, or joint involvement. There was a moderately loud murmur typical of mitral regurgitation. No diastolic murmur was heard. Along the left border of the sternum was a well defined pericardial rub. We advised that she be started on cortisone in somewhat larger doses than would be given usually. There was no improvement from cortisone and within ten days signs of hypercortisonism (weight gain and rising blood pressure) appeared. The cardiac rub persisted and there was no lessening of the fever or prostration.

After two weeks the cortisone was stopped and aspirin was given, six grams daily. No beneficial effect occurred in a 12 day trial of this therapy. Rheumatic fever of the severity manifested in this patient nearly always causes congestive heart failure. This feared development occurred weeks after admission in the manner in which we see congestive failure appear in children with active rheumatic fever. The liver became enlarged and a gallop rhythm heard not at the cardiac apex but along the upper left sternum was noted. There had not been any orthopnea or notable dyspnea. Pulmonary rales have been absent. This clinical picture is characteristic of congestive failure in children with active rheumatic fever.

As is our custom diuretic therapy was instituted with theocalcin, one gram three times a day, and mercuhydrin, 1 cc intramuscularly once or twice a week as needed. While the congestive failure did not increase it

did not diminish except for a few hours. In the face of an ever worsening picture, completely resistant rheumatic fever productive of considerable congestive failure, digitalization should be considered. As a rule it fails to help but occasionally striking benefit does occur. Three weeks after admission four cc (0.8 mg) of cedilanid was given intravenously in one dose. Digitalis effect has been maintained by 0.5 gm. of the whole leaf daily. Within twelve hours of starting digitalis the first definite improvement since the onset of this girl's illness was noted. Her liver diminished in size by 50 per cent and there was a distinct decrease in her toxic appearance. In addition, as can be seen on the temperature chart, her fever has disappeared. I have no clear explanation for this antipyretic effect. We are seeing an unusual but clearcut, rather striking benefit due to digitalis.

The management of rheumatic fever has not changed materially in the last decade or more. As with most diseases of unknown cause the therapy of rheumatic fever is empirical. This continues to be the case despite the advent in 1948 of ACTH and cortisone. Just four years ago Dr. Leyva, assisted by the cardiac service, treated the first rheumatic fever patient in this hospital with ACTH and cortisone. Since that time we have treated nearly fifty rheumatic fever patients with these new drugs. Most of this group have been treated at the Rheumatic Fever Unit of the Crippled Children's Service at the District General Hospital. The case histories of the first 24 patients in this group have been analyzed by Dr. John L. Siddoway, Jr. and myself. Seven of these patients seemed to me to be very definitely benefited by cortisone and ACTH. Six of the group were rather like this patient who is under discussion and in none of these was there any beneficial effect. I had it in mind to publish these experiences until I learned at a special meeting of the Council on Rheumatic Fever and Congenital Heart Disease in Chicago in 1952 of the preliminary analyses of a group of 350 rheumatic fever patients treated with ACTH, cortisone, and aspirin. This wonderfully planned and carried-out study sponsored by the council, which has now been completed with 750 patients included in the series, revealed that those patients treated with ACTH and cortisone did no better in any respect than those treated with aspirin. The progress of the disease and the amount of heart damage were parallel for all three drugs.

In the course displayed by the patient who is being considered in this discussion, we have a good example of the apparent failure of cortisone and salicylates given in large dosage to ameliorate any phase of the disease. The first favorable turn in the course of her illness has come with the use of digitalis. This good effect may be only temporary, but I think the gain must be attributed to digitalis. We do not regularly use digitalis in the treatment of congestive failure due to active rheumatic fever because it infrequently benefits the patient and there is a high risk of producing

auricular fibrillation. When this arrhythmia appears in these patients the ventricular rate, is, as a rule, remarkably fast, 160 to 180 per minute, which in turn brings about very undesirable physiologic effects. However, in a desperate situation which seemed to be proceeding to a fatal termination, the use of digitalis, despite some risk, is, I feel sure, warranted. This girl has been given an antibiotic daily from the time of her admission to the hospital to protect her against intercurrent respiratory infection. For a time she was on high dosage of penicillin and terramycin because of some suspicion on the part of the medical service that she had bacterial endocarditis rather than rheumatic fever.

*Addendum.* It is now three weeks since this patient was presented at the clinical conference. She has improved steadily in this time. She is free of complaint and has a good appetite. There has been no evidence of congestive heart failure for more than two weeks. We are planning to discharge her to a convalescent home after two or three more weeks of observation.

DISCUSSION

Proctor Harvey, M.D.

There is very little to add to Dr. Walsh's discussion. All of us have seen cases of particularly acute rheumatic fever where we would like to give more cortisone. Of course, we have all seen good results with the hormones. Cortisone could be called "a super aspirin." Often, perhaps, we are treating ourselves. When we see a patient who is doing poorly we want to do something but we want to be certain that we are doing the right thing. This case exemplifies excellent management. Every type of treatment was used here. The withholding of digitalis until signs of decompensation occurred is an example. I think all of us have been in that position where the patient is doing very poorly, and we feel that we must do something. As a rule digitalis does help them.

I would like to add one thing which is I think pertinent to this case. What will be the management of this case in the future? Much work has been done on the prevention of rheumatic fever, but the recurrence of rheumatic fever can be cut down many times by the use of oral tablets of penicillin and bicillin. That aspect is one which has not been emphasized enough. All dental work and all sore throats in patients of this type should be covered by antibiotics. We need not wait to get cultures. Recent studies show that we should keep up the treatment of streptococcal sore throats with antibiotics for at least ten days.

## CLINICO-PATHOLOGICAL CONFERENCE

Monday, September 21, 1953

Directed by: E. Clarence Rice, M.D.
Assisted by: Roger Bergstrom, M.D.
By Invitation: O. H. Fulcher, M.D.

A four year old white female was admitted to Children's Hospital with the following history:

Six weeks before admission she developed a sore throat and a partial paralysis of the right face, arm and leg. The right corner of the mouth could not be "pulled up" and the right arm and leg were weak. The latter caused the child to limp. The sore throat cleared promptly and the paralysis gradually improved but did not disappear completely.

Twelve days before admission while eating lunch, the patient complained of inability to see, and when put to bed, she thrashed about wildly, chewed the bed linen, screamed with pain, and lost consciousness. Her right arm and leg became rigid and were held in extension. Later it was noticed that the extremities on the right occasionally flexed slowly but usually were "straight and stiff." The child regained consciousness but was very "dull" mentally and ate very little. In the next few days she became more and more stuporous and lost consciousness completely the day before admission. At this time hospitalization was advised.

The patient was delivered at term at home with the aid of forceps and weighed seven and one half pounds. Her head was misshapen at birth but soon appeared normal. She had a normal nutrition and development and there was a history of whooping cough at five weeks, chicken pox at two years and measles at three years. During the first year of life she was seen at Episcopal Hospital where a diagnosis of xerosis of the right eye was made and vitamin A recommended. No accidents preceded the present illness except for a fall on the back of the head without loss of consciousness one year previously.

The family history was non-contributory.

Physical examination on admission revealed a pale, comatose child. Occasional purposeless movements of the upper extremities, particularly the right, were observed. The temperature was 102.6° rectally and the systolic blood pressure was 110 millimeters. Pulse and respiratory rates were not recorded. There were numerous petechiae on the palms and soles and over the body. There were larger erythematous areas which did not blanch on pressure. These were thought to be hemorrhagic.

The head appeared normal in size and shape. The pupils were semi-dilated and fixed. The right pupil was largely occluded by a dense corneal scar. The retinal vessels seemed dilated and tortuous and the disk was blurred with an exudate but there was no obvious hemorrhage. The left fundus was filled with large and small hemorrhages and exudate. The disk was almost obliterated, there were three diopters of choking and the veins were full.

The ear drums were normal except for several petechiae. The mouth and throat could not be examined because the jaws were shut tightly. Heart and lungs were normal, no masses or organs could be felt in the abdomen, and there was no lymphadenopathy. The lower extremities were rigid and extended. The deep reflexes were exaggerated and the Babinski was positive bilaterally. The Kernig sign was suggestively positive and the Brudzinski was negative. Hemogram revealed a hemoglobin of 11 gm. per cent, erythrocytes 3,080 and leukocytes 10,800 with 87 per cent neu-

trophiles. 20 cc. of clear colorless fluid was removed from the lumbar spine under apparently increased pressure. It was normal in cellular and chemical content.

Shortly after the spinal puncture numerous red patches appeared over the body and the patient became cyanotic and stopped breathing. The pulse was then noted to be rapid and thready. She was given artificial respiration and stimulants but failed to respond and was pronounced dead four hours after admission.

DISCUSSION

O. H. Fulcher, M.D.

Dr. Bergstrom has presented a most interesting case history. The early symptom of a sore throat associated with partial right hemiplegia would certainly suggest an infectious process. I would like to know which developed first; the sore throat or the partial right hemiplegia? If the hemiplegia existed prior to, or developed concomitantly with the onset of the sore throat, then it probably had a separate etiology. The incidence of sore throat among children is so frequent that it could likely be incidental. On the other hand if the onset of sore throat preceded the beginning of the right hemiplegia by several days, then there could exist a causal relationship. I wish very much that the records had been adequate to permit a better analysis. Since the paralysis improved after the sore throat disappeared, one could interpret this fact as further evidence of a causal relationship. I hesitate to stress this point too strongly because so frequently it appears to me that the case report has been selected because of the presence of a red herring.

There is a complete blank in the history for about four weeks. Then rather suddenly the symptoms and signs changed. The patient became very restless. This restlessness probably represented a change in the state of consciousness. She did become unconscious and the extremities became rigid which would denote cortical stimulation of the left motor area. As one attempts to solve the diagnostic puzzle at this stage, one could interpret the onset of symptoms and signs as a left cerebritis secondary to an infection of the throat. Then during the four weeks of no records, the cerebritis developed into a circumscribed abscess. About twelve days before admission there could have been a spontaneous rupture of the abscess with loss of consciousness and severe irritation of the left motor cortex. The records do not show just how long the child was unconscious. This information is desirable. Gradually she became unconscious again which could be interpreted as a progression of the inflammatory process.

The history of the birth of the child and of infancy extending to the onset of the present illness does not appear to contribute to the problem of developing a diagnosis. A fall one year previously will be remembered if subsequent analysis could attribute the findings as compatible with those produced by a chronic subdural hematoma.

The physical examination revealed the child to be comatose and to have a temperature of 102.6 degrees rectally. It is possible of course that the temperature could result from dehydration but it could more likely be caused by an infection. The petechiae and erythematous areas would be compatible with an infectious process. Also one must remember that the child probably had poor supervision at home or else earlier hospitalization would have occurred. Hence, the great likelihood of dehydration and poor hygenic care of the skin. The choked disc on the left demonstrated beyond doubt the presence of increased intracranial pressure. It is assumed that the dense corneal scar rendered impossible the inspection of the right fundus. The presence of papilloedema constituted a contraindication for spinal puncture. The failure to respect this contraindication constitutes always a hazard of the gravest type as was unhappily demonstrated with this patient.

The clear spinal fluid with normal cellular and chemical contents does not comply with the expected findings if a brain abscess existed. The normal spinal fluid is the first jolt to the concept of a brain abscess which I have been developing.

The leucocytes and the percentage of neutrophiles are compatible with a low grade infectious reaction that would be caused by a circumscribed abscess.

In summary, I believe that the patient was moribund upon admission but that death was precipitated by the spinal puncture which permitted the medulla to herniate through the foramen magnum because of the much increased intracranial pressure.

Much additional information concerning the history is desired but cannot be obtained. An analysis of the data presented will permit no conclusive diagnosis. I think that the history and the findings will most closely warrant the impression of a left cerebritis secondary to an infected throat; that the cerebritis developed into a circumscribed abscess of the left fronto-parietal area. The chief positive objection to this conclusion is the existence of normal spinal fluid.

### PATHOLOGICAL DISCUSSION

Jerome Bernstein, M.D.

The post mortem examination was limited to the head. The bones of the skull were slightly thinner on the right than on the left. The dura appeared normal and there was no evidence of subdural effusion.

The brain substance was firm, slightly dry and the convolutions were moderately flattened. There was an area in the brain stem just superior to the left of the pons, which was very thin, measuring 1.5 x 0.75 cm, and covered only by a thin layer of pia mater.

Gross examination of the brain after fixation showed the right lateral ventricle to be dilated and to contain blood tinged fluid. The left lateral ventricle was compressed from below and laterally by a large tumour mass, the tip of which could be seen projecting near the juncture of the crus and the temporal lobe. From that point it extended upward and outward, occupying a space 7.0 x 4.5 x 3.5 cm.

The tumour mass appeared to arise from a point anterior to and above the fourth ventricle. It occupied the greater portion of the left parital lobe and a portion of the left occipital lobe. The mass was relatively soft and fluctuant being dark red and hemorrhagic in appearance with no definite capsule.

Microscopically, the specimen is made up of cerebral tissue in which there are many vessels, irregular in outline and arrangement and frequently lined by broad somewhat irregular endothelial cells. There are a number of areas of hemorrhage. Plasma cells, fatbearing cells and phagocytic cells were noted in the tissue. The lesion is representative of cerebral hemorrhage

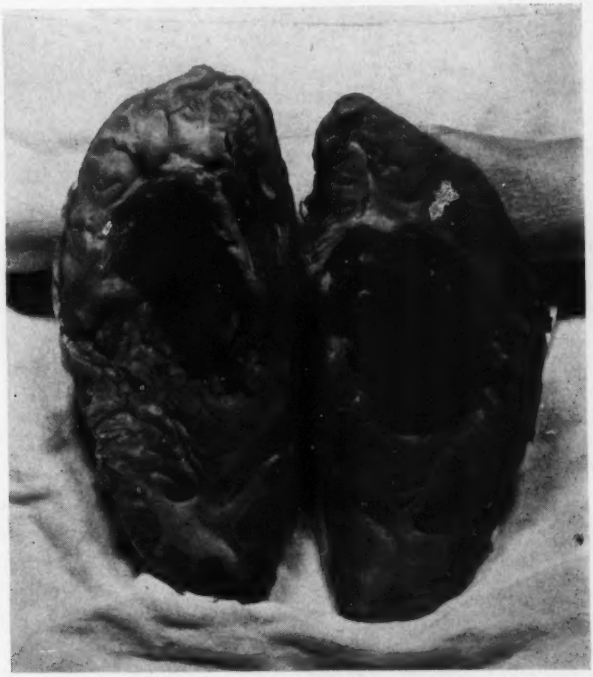

FIG. 1. Left hemisphere showing hemorrhage into the cortex compressing the ventricles.

apparently arising from an area of unusual vascularity and is believed to be representative of a hemangioma. No evidence of malignancy is seen.

Pathological Diagnosis: Hemangioma with hemorrhage and inflammatory reaction.

E. Clarence Rice, M.D.

Hemangiomas of the meninges and brain are reported to be most commonly seen in the younger age group of patients, but have not been seen with any frequency at this hospital. The meninges are most commonly involved. When present in the brains of children, the cerebellum is the most common site, the lesion sometimes being cystic. Multiple organs may be involved. A familial relationship has been reported in some patients.